Oxalate Nephropathy Due to High Oxalate Vegan Diet

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Key Words: Oxalate nephropathy, acute kidney injury, dietary hyperoxaluria

Abstract

Oxalate nephropathy is associated with progressive renal injury due to intratubular precipitation of calcium oxalate leading to renal failure. It can be caused by primary or secondary hyperoxaluria. Secondary hyperoxaluria can occur with excessive intake of oxalate (or substances that metabolize to oxalate), increased oxalate absorption or decreased oxalate excretion. The following article is a case study presentation of a patient with chronic kidney disease secondary to oxalate nephropathy after excessive consumption of high oxalate foods.

The case reviewed in this article is that of a 52 year old male with worsening kidney function over a period of one year. His creatinine increased progressively and kidney biopsy results showed significant amounts of oxalate crystals. Serum and urine oxalate levels were elevated at 19.9(<1.8) µmol/L and 132(9.7-40.5) mg/24hr, respectively. Genetic testing for primary hyperoxaluria was negative. The patient’s only identified risk factor for oxalate nephropathy was a vegan diet with excessive dietary intake of foods containing high oxalate levels.

Intake of oxalate rich foods may potentially precipitate acute renal failure, especially in patients with mild to moderate chronic kidney disease. In our case study, the patient’s only risk factor for hyperoxaluria was high dietary oxalate intake. Awareness of high oxalate content foods may help prevent kidney damage associated with oxalate nephropathy.

Introduction

Oxalate is the salt-forming ion of oxalic acid, which is an end-product of plant and animal metabolism (Figure 1). This ion binds easily to calcium, and forms a nearly insoluble salt that tends to crystallize (1,2). Oxalate molecules are filtered by the kidneys through the glomerulus. When there is an increased content of urinary oxalate beyond dissolution, the complex formed with calcium supersaturates the urine causing precipitation. Acute oxalate nephropathy is caused by deposits of oxalate crystals in the tubules and

-- Continued on page 3.
Hannah Sobol, RDN, CSR
Editor

As things are warming up here in Phoenix, AZ it is always helpful to have great reading material to entertain your brain while staying indoors and out of the heat! The summer issue of the Forum is no exception and will certainly stimulate your renal dietitian mind while offering 3.0 CPEUs.

The feature article discusses the connection between a high oxalate vegan diet and oxalate nephropathy in chronic kidney disease (CKD). In the Use of Diabetes Medication in the Patient with CKD article, the author gives a wonderful summary of which diabetic medications are preferred in CKD, as well as which drugs need dose adjustments and which classes of medications have evidence to support their use in CKD. The next two following articles give perspective on practicing mindfulness in taking care of one’s diabetes and insulin pump use for dialysis patients. We all need reminders to stop and be more mindful and aware of our surroundings, our thoughts and emotions and this article was a helpful reminder of what our diabetic patients may be struggling with.

You will enjoy Rosa Hand’s research project poster and write up, funded by RPG, focusing on RDN staffing ratios in dialysis centers and how this relates to patient outcomes. Be sure to check out the latest app review of The 7 Day Food Journal Challenge app. Also, included in this Forum is another Century Anniversary piece by Mary Kay Hensley, MS, RD, CSR highlighting what current RDNs foresee in the future of our profession as we look toward the next 100 years.

My hope for the future of our profession is that the healthcare industry will see the value of early nutrition education in both CKD and diabetes and that access to RDN’s with expertise in these areas will continue to grow.

In closing I would like to say thank you to my co-editors Michelle and Stacey, and managing editor Desiree de Waal, for all their support and insight. As always, a special thanks to our test question writer Amy. Enjoy reading!
results in inflammation, interstitial fibrosis, and progressive renal injury. However, in patients with decreased kidney function who present with impaired renal clearance, serum oxalate levels may increase leading to tissue crystal formation in uremic patients (3). Autopsies in patients with end stage kidney disease have shown a correlation between serum oxalate levels and severity of crystal deposition in the heart and kidneys (4). The incidence and severity of this type of secondary oxalosis was found to be related to the duration of renal failure.

About 75% of all kidney stones are primarily composed of calcium oxalate. The pathogenesis of stone formation consists of crystal nucleus formation, followed by growth and aggregation (5). Once a calcium oxalate crystal has formed, it starts to grow by further deposition of crystal components. If urinary oxalate concentration remains elevated, this may lead to stone formation (6). This mechanism has been confirmed by in vitro studies performed by Lieske et al. showing intracellular crystal engulfment as a possible pathway of stone formation (7). There are several promoters of stone formation (Table 1), such as low urine volume and pH, as well as high sodium, calcium and oxalate content in urine. In contrast, several substances have been shown to inhibit stone formation, including citrate and magnesium (5).

Etiology of Hyperoxaluria

Several etiologies for hyperoxaluria have been identified, including endogenous and exogenous causes such as intrinsic overproduction, increased ingestion and/or absorption of oxalate or its metabolites (i.e. oxalic acid, ascorbic acid, glycolic acid). Increased oxalate excretion after renal transplantation in patients with primary hyperoxaluria has also been described (8). In some patients the cause of hyperoxaluria could be multi-factorial (9).

**Primary Hyperoxaluria**

Genetic disorders causing endogenous overproduction of oxalate are referred to as primary hyperoxaluria (PH). This group of rare autosomal recessive inherited diseases has been associated with increased urinary excretion of oxalate. In type I PH, defective hepatic enzyme activity of alanine glyoxalate aminotransferase (AGT) may lead to high content of calcium oxalate in blood deposits in the heart, bone marrow and kidney. Urinary oxalate concentration may be as high as 300 mg/24 hrs. Molecular analysis showing mutations of the AGXT gene (PH, type I), GRHPR gene (PH, type II) or HOGA1 gene (PH, type III) may confirm diagnosis in some patients (10, 11).

**Table 1. Risk factors for stone formation***

<table>
<thead>
<tr>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low urine volume</td>
</tr>
<tr>
<td>Low fluid intake</td>
</tr>
<tr>
<td>High oxalate intake</td>
</tr>
<tr>
<td>High sodium intake</td>
</tr>
<tr>
<td>High protein intake</td>
</tr>
<tr>
<td>High vitamin C intake</td>
</tr>
<tr>
<td>High urinary calcium</td>
</tr>
<tr>
<td>Low urinary citrate</td>
</tr>
<tr>
<td>Urine pH</td>
</tr>
<tr>
<td>Family history of kidney stones</td>
</tr>
<tr>
<td>Frequent urinary tract infections</td>
</tr>
<tr>
<td>Anatomic abnormalities such as</td>
</tr>
<tr>
<td>• horseshoe kidney, medullary sponge kidney</td>
</tr>
<tr>
<td>Malabsorption conditions such as</td>
</tr>
<tr>
<td>• Crohn’s disease, gastric bypass surgery</td>
</tr>
</tbody>
</table>

*References: 5,6,13,14,33,35
Secondary Causes of Hyperoxaluria

In general, the human body is able to excrete excess intake of oxalate from our diets. However, when there is malabsorption associated with bowel disease such as Crohn’s disease, celiac disease, short bowel syndrome or gastric bypass surgery, there is an increased absorption of colonic oxalate (12). Because of fat malabsorption, a high content of lipids can remain in the gut. Lipids bind available calcium, thereby decreasing calcium’s availability to generate calcium oxalate complexes, and resulting in excessive oxalate absorption (13). Orlistat, a lipase inhibitor used in weight reduction therapy, has also been shown to increase urinary oxalate, increasing the risk of kidney stones and nephrocalcinosis (14).

Ethylene glycol ingestion has been associated with oxalate nephropathy (15). Ethylene glycol is an odorless and sweet-tasting alcohol found in antifreeze. Upon intentional or unintentional ingestion, this toxin degrades to oxalic acid and precipitates in the kidneys, causing acute tubular damage. Severe kidney injury can cause irreversible damage that may be life-threatening, requiring treatment with dialysis (16).

Multiple factors were found to play a role in calcium oxalate crystal formation in the kidneys of transplanted patients, including malabsorption and modification of intestinal flora due to antibiotic use. During the first few weeks after kidney transplantation, the allograft is able to excrete accumulated oxalate in a short period of time, promoting deposition and allograft damage. Calcium oxalate deposits may be seen in kidney allograft biopsies within 3 weeks after transplant. This phenomenon is associated with poor allograft survival (17). In one retrospective study of 65 patients with biopsy-proven renal calcium oxalate crystals, 17% of the cases had a history of kidney transplantation as a single risk factor, and 12% had a second risk factor, primarily a high oxalate diet (9). Oxalate nephropathy following non-renal solid organ transplantation (i.e. lung, lung-liver) has also been described (18).

Dietary Hyperoxaluria

Excessive dietary intake of oxalate may lead to urinary hyperoxaluria. Oxalate rich diets contribute to a urinary oxalate excretion ranging from 24 to 42 mg/day for a 2,500 calorie diet. Furthermore, if the diet is low in calcium, oxalate levels in the urine may increase to 53 mg/day (19).

Natural sources of oxalate in our diet are varied. These include almonds, peanuts, soybeans and other legumes, some leafy greens such as spinach and rhubarb, chocolate, and tofu, among others (20-24,29) (Table 2). Star fruit has also been identified as a source of high oxalate content (25). Fang et al. showed that rats fed with star fruit juice developed acute kidney injury and their renal biopsies showed typical changes of oxalate nephropathy (26). Cases associated with black iced tea and oxalate-rich fruit and vegetable juicing as causes of oxalate nephropathy have been reported (9,27). Nuts have been associated with acute oxalate nephropathy (28). Of the tree nuts, almonds have been found to have the highest content of oxalate (Table 3) (20). Although high dietary oxalate alone has not been proven to lead to hyperoxaluria, the increased intestinal absorption of dietary oxalate has been associated with a higher risk of stone formation (19).

Oxalate is also the end product of vitamin C metabolism. A significant increase in urinary oxalate content ranging from 48 to 79% has been reported in calcium-stone formers after supplementation with 1-2 g of vitamin C (29,30). The use of over-the-counter vitamin C supplementation may lead to potentially toxic consequences causing irreversible kidney damage, even in previously healthy individuals (31).

Table 3. Oxalate content in nuts*

<table>
<thead>
<tr>
<th>Food Item</th>
<th>Oxalate (mg/ per 100 g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almonds</td>
<td>469</td>
</tr>
<tr>
<td>Soy nuts</td>
<td>392</td>
</tr>
<tr>
<td>Cashews</td>
<td>262</td>
</tr>
<tr>
<td>Hazelnuts</td>
<td>222</td>
</tr>
<tr>
<td>Pine nuts</td>
<td>198</td>
</tr>
<tr>
<td>Peanuts</td>
<td>140</td>
</tr>
<tr>
<td>Walnuts</td>
<td>74</td>
</tr>
<tr>
<td>Pecans</td>
<td>64</td>
</tr>
<tr>
<td>Pistachio</td>
<td>49</td>
</tr>
<tr>
<td>Macadamia nuts</td>
<td>42</td>
</tr>
</tbody>
</table>

*References: 21, 22

Case Study

A 52 year-old male was referred to our nephrology clinic for evaluation of progressively worsening chronic kidney disease (CKD). His past medical history was remarkable for migraine headaches treated prophylactically with verapamil and, as needed, use of NSAIDs. The patient was not taking any other medications or over-the-counter supplements. There was no family history of kidney disease and no previous personal history of kidney stones, gastric surgeries, diarrhea, or other urinary tract symptoms. His creatinine increased from a baseline of 1.2 mg/dL to 1.4 mg/dL during a period of 4 years, and his CKD was thought to be caused by chronic NSAID use in the setting of unremarkable urinalysis (Table 4). Therefore, NSAIDs were discontinued.

Table 2. High Oxalate Content Foods (greater than 100 mg/100g)*

<table>
<thead>
<tr>
<th>High Oxalate Content Foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almonds</td>
</tr>
<tr>
<td>Peanuts</td>
</tr>
<tr>
<td>Soy protein</td>
</tr>
<tr>
<td>Beets (root)</td>
</tr>
<tr>
<td>Rhubarb</td>
</tr>
<tr>
<td>Star fruit (Carambola)</td>
</tr>
<tr>
<td>Cashews</td>
</tr>
<tr>
<td>Sesame seeds (Tahini)</td>
</tr>
<tr>
<td>Swiss chard</td>
</tr>
<tr>
<td>Dark chocolate (large intake)</td>
</tr>
<tr>
<td>Spinach</td>
</tr>
<tr>
<td>Tofu</td>
</tr>
</tbody>
</table>

*References: 21-25,29
 FEATURE ARTICLE

Table 4. Patient’s urinalysis

<table>
<thead>
<tr>
<th>Urinalysis</th>
<th>Value</th>
<th>Normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>4.7</td>
<td>4.5-8.0</td>
</tr>
<tr>
<td>Osmolality</td>
<td>393 mOsm/Kg</td>
<td>150-1150 mOsm/Kg</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Protein</td>
<td>4 mg/dL</td>
<td>&lt; 22 mg/dL</td>
</tr>
<tr>
<td>Microscopy</td>
<td>Normal</td>
<td>Normal (no cells or crystals seen)</td>
</tr>
<tr>
<td>Albumin/Creatinine</td>
<td>&lt; 7 mg/g</td>
<td>&lt; 17 mg/g</td>
</tr>
</tbody>
</table>

Table 5. Patient’s plasma work up

<table>
<thead>
<tr>
<th>Plasma sample</th>
<th>Value</th>
<th>Normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>140 mmol/L</td>
<td>135-145 mmol/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.3 mmol/L</td>
<td>3.6-5.2 mmol/L</td>
</tr>
<tr>
<td>Chloride</td>
<td>104 mmol/L</td>
<td>98-107 mmol/L</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>25 mmol/L</td>
<td>22-26 mmol/L</td>
</tr>
<tr>
<td>Blood urea nitrogen</td>
<td>41 mg/dL</td>
<td>8-24 mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>2.5 mg/dL</td>
<td>0.8-1.3 mg/dL</td>
</tr>
<tr>
<td>Total calcium</td>
<td>9.8 mg/dL</td>
<td>8.9-10.1 mg/dL</td>
</tr>
<tr>
<td>Uric acid</td>
<td>8.2 mg/dL</td>
<td>3.5 – 8 mg/dL</td>
</tr>
<tr>
<td>Albumin</td>
<td>4.8 g/dL</td>
<td>3.5 – 5 mg/dL</td>
</tr>
<tr>
<td>Oxalate</td>
<td>19.9 µmol/L</td>
<td>&lt;1.8 µmol/L</td>
</tr>
</tbody>
</table>

However, despite discontinuation of NSAIDs, his creatinine continued to worsen from 1.4 mg/dL to 2.1 mg/dL over a period of 11 months. Upon presentation to our clinic, his physical exam was unremarkable; his body mass index was 23. His creatinine was further elevated to 2.5 mg/dL and blood urea nitrogen was 41 mg/dL. Laboratory investigation showed anemia with a hemoglobin of 11.5 g/dL, a white blood cell count of 5.5 x10^9/L, and normal platelets. His iron stores were slightly elevated: iron level 158 mcg/dL (normal 60-150 mcg/dL), ferritin 383 mcg/L (normal 20-50%); he had recently received IV iron supplementation for anemia. Serum electrolytes, calcium, phosphorus, and bicarbonate were within normal limits; only uric acid was mildly elevated at 8.2 mg/dL. (Table 5). Rheumatological work-up, including extractable nuclear antibodies panel, vasculitis and a hepatitis panel were all negative. C3-C4 complements were normal. No monoclonal protein was identified. A repeated urine sediment was again unremarkable and the renal ultrasound showed normal sized kidneys with increased parenchymal echogenicity, but no hydropnephrosis or other structural abnormalities. The renal artery doppler was normal. Due to the unexplained cause of his renal dysfunction, a kidney biopsy was performed. The biopsy showed a significant amount of intratubular polarizable oxalate crystals (Figure 2) with associated interstitial fibrosis. The results prompted evaluation of serum oxalate level and it was elevated at 19.9 µmol/L (normal <1.8µmol/L). A 24hr urine oxalate was also elevated at 132.9 mg (normal 9.7-40.5 mg/24 hours). His 24 hour proteinuria was 77 mg/dL (Table 6). Genetic testing for the three forms of primary hyperoxaluria (PH) was negative. The patient’s only risk factor for oxalate nephropathy was a vegan diet with intake of high oxalate foods (Table 7). His daily breakfast was rich in oxalate containing oatmeal with chia and flax seeds and raisins or prunes. For lunch, he usually had vegetables, including a spinach salad with dry beans, sesame seeds and hummus. Dinner often consisted of a salad with beets and tofu. He was not taking any calcium supplements, and his dairy intake consisted of one glass of low-fat milk per day. His daily fluid intake was about 1.5 to 2L of water. His diet was also rich in a variety of berries, almonds, and dark chocolate. His almond consumption was approximately 3 ounces (85 mg) per day, with an estimated oxalate content of 398 mg per day. In the absence of other known risk factors, the diagnosis of oxalate nephropathy due to high oxalate dietary intake was made. He was referred to a renal dietitian nutritionist who educated him about the average oxalate content of his current diet and recommended portion sizes for high oxalate content foods. He was also advised to increase his fluid intake to 3L per day, and to take calcium citrate with meals. Two months after these changes were made his kidney function improved, shown by a creatinine decrease from 2.5 mg/dL to 2.1 mg/dL.

Table 6. Patient’s 24-hour urine collection

<table>
<thead>
<tr>
<th>Urinary concentration</th>
<th>Value</th>
<th>Normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>116 mmol</td>
<td>41-227 mmol</td>
</tr>
<tr>
<td>Potassium</td>
<td>121 mmol</td>
<td>17-77 mmol</td>
</tr>
<tr>
<td>Calcium</td>
<td>52 mg</td>
<td>25-300 mg</td>
</tr>
<tr>
<td>Oxalate</td>
<td>132.9 mg</td>
<td>9.7-40.5 mg</td>
</tr>
<tr>
<td>Citrate</td>
<td>223 mg</td>
<td>370-1100 mg</td>
</tr>
<tr>
<td>Uric acid</td>
<td>371 mg</td>
<td>&lt;750 mg</td>
</tr>
<tr>
<td>Total protein</td>
<td>77 mg</td>
<td>&lt;167 mg</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1669 mg</td>
<td>15-25 mg/kg</td>
</tr>
<tr>
<td>Volume</td>
<td>2337 ml</td>
<td>1000-5000 ml</td>
</tr>
</tbody>
</table>

Table 7. Example of a typical day based on patient’s diet journal

<table>
<thead>
<tr>
<th>Breakfast</th>
<th>Oatmeal with chia and flax seeds</th>
<th>Raisins or prunes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lunch</td>
<td>Spinach salad with beans</td>
<td>Sesame seeds and hummus</td>
</tr>
<tr>
<td>Dinner</td>
<td>Lettuce salad with beets and tofu</td>
<td></td>
</tr>
<tr>
<td>Snacks</td>
<td>1 ounce of almonds three times per day</td>
<td></td>
</tr>
</tbody>
</table>
large oxalate intake of about 750 mg per day (36). Probiotics containing anaerobic bacteria, such as Lactobacillus acidophilus, have shown to prevent intestinal oxalate absorption and therefore decrease oxalate urinary excretion (37,38).

Additional therapies have been studied to treat secondary hyperoxaluria, but have not yet been implemented as part of standard management. ALLN-177 is an oral formulation of a recombinant form of a microbial enzyme that degrades dietary oxalate in the gut. It has been shown to decrease urinary oxalate excretion in healthy volunteers; however more trials to determine its safety are required before its accepted use (39).

Conclusion
We presented a case of oxalate nephropathy associated with high oxalate consumption in a patient with an oxalate rich vegetarian diet. Our patient had no other risk factors for hyperoxaluria. Identification of calcium oxalate crystals in a kidney biopsy should prompt further detailed dietary history. Intake of oxalate rich foods may potentially precipitate acute renal failure, especially in patients with mild to moderate CKD. A referral to a dietitian can help assess dietary risk factors for oxalate nephropathy. Decreased consumption of these foods may help prevent kidney damage associated with oxalate nephropathy.

Conflict of Interest
The authors do not have any financial or non-financial potential conflicts of interest.

References

Hyperoxaluria Management
High fluid intake of more than 2L per day and urine alkalinization have been the main therapy to prevent stone formation (32). Vitamin B6 (pyridoxine) has been recommended as part of the management for PH type I, with the purpose to decrease urine oxalate excretion (33).

For patients with secondary hyperoxaluria, diet plays a major role in reducing the risk of oxalate nephropathy. Avoiding oxalate rich foods helps to reduce hyperoxaluria. In individuals at higher risk, especially those following a strict vegan diet or with known risk factors for kidney disease (i.e. history of kidney stones, diabetes), other measures, such as diet changes, may be beneficial.

Individuals who follow a DASH-style diet, consisting of a high content of fruits and vegetables, moderate intake of low-fat dairy products, and low content of animal protein, may tend to have a higher oxalate intake when compared to other types of diets. Although this may increase the urinary oxalate, and therefore increase the risk for calcium oxalate stone formation, high intake of fruits and vegetables may also help increase urinary citrate, known to be an important inhibitor of calcium stones (32,34). Furthermore, a direct comparison done between the DASH and low-oxalate diets showed a decrease of calcium oxalate supersaturation in those following a DASH diet, associated with higher urinary pH and increased urinary magnesium and citrate excretion (35). Unfortunately our patient had a low urinary citrate despite the daily consumption of fruits and vegetables in his diet.

Ingestion of the recommended dietary allowance of calcium in the diet (i.e. 1000 mg of calcium per day) has been shown to decrease the risk of stone formation in those who have a


Use of Diabetes Medications in the Patient With Chronic Kidney Disease

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Objectives
1. Summarize the evidence supporting the use of various classes of medications in patients with CKD.
2. Describe which antidiabetic medications need dosing adjustments when prescribed to a patient with CKD.
3. Discuss which antidiabetic agents are preferred within each class of medications based on their efficacy and safety when used in patients with CKD.

Abstract
A large body of evidence exists describing the utility of antidiabetic agent use in patients with chronic kidney disease (CKD). Studies have evaluated utilization of all agents in various stages of renal impairment and clinical decisions should take into account pharmacokinetic variability seen with use of these agents in renally impaired patients. Dosage reduction recommendations and limitations for use exist based upon these outcomes.

Introduction
Antidiabetic agents must be carefully selected for patients with varying degrees of CKD. Due to the high incidence of renal impairment in those who have diabetes mellitus (DM), it is crucial to continually monitor kidney function and adjust medication doses in accordance with renal impairment. Monitoring of blood glucose as well as kidney function should be ongoing. A significant body of literature has been published describing the effects of CKD on the patient with DM that clinicians should understand when considering the effects of a patient’s pharmacologic regimen.

Literature Review
DPP-4 Inhibitors
Dipeptidyl peptidase-4 (DPP-4) inhibitors increase endogenous exposure to native glucagon-like peptide-1 (GLP-1) through inhibition of the enzyme responsible for its degradation (DPP-4). They have a low incidence of hypoglycemia or other adverse effects while demonstrating weight neutrality. Due to their extensive renal clearance, dose adjustments are recommended (1).

Sitagliptin (Januvia®) has demonstrated up to a 3.8-fold increase in availability in patients with severe renal impairment. Because of this, manufacturer recommendations suggest decreasing the dose from 100mg daily to 50mg daily if creatinine clearance (CrCl) ≤ 50mL/min and further to 25mg daily if CrCl is less than 30mL/min (2).

Alternatively, a second DPP-4 inhibitor, saxagliptin (Onglyza®) is metabolized via CYP450 enzymes to an active metabolite, which is, in turn, eliminated renally. Studies have demonstrated that patients with renal impairment receiving a 2.5 mg dose (half of the usually prescribed dose) had a significant reduction in hemoglobin A1c (HbA1c). Due to these findings, a decrease in the daily dose from 5mg to 2.5mg is recommended for saxagliptin in those individuals with a CrCl 50 mL/min or less (3).

Fewer studies exist with alogliptin (Nesina®), but a review of several pharmacokinetic studies confirmed the need to dose adjust alogliptin in patients with CKD. Alogliptin was found to have similar drug accumulation to sitagliptin in patients with renal impairment. Alogliptin maintains efficacy in lowering HbA1c but should be reduced to 12.5mg for CrCl of 30-59 mL/min and 6.25mg for CrCl of less than 30 mL/min (4,5).

The exception to the commonly high renal clearance of the DPP-4 inhibitors is linagliptin (Tradjenta®), with only 5% of the oral dose being eliminated renally. The renal effects on clearance and long-term exposure are minor, therefore, requiring no dose adjustment in the presence of CKD (6).

Beyond the convenience of not needing a dosage adjustment with linagliptin use, data exists on a possible benefit in reducing albuminuria. A pooled analysis of four randomized, double-blind, placebo controlled studies totaling 217 patients with type 2 diabetes and albuminuria while receiving stable renin-angiotensin-aldosterone system (RAAS) inhibitors were randomized to either linagliptin 5mg daily or placebo. Albuminuria, measured as urinary albumin-to-creatinine ratio (UACR) of 30-3,000 mg/g creatinine, was significantly reduced from baseline by 28% in patients receiving linagliptin. This reduction in albuminuria is associated with a decreased risk of progression of renal impairment, including end-stage renal disease (ESRD). Although the mechanism is not fully understood, the potential albuminuria-lowering effect of linagliptin has been seen in previous studies as well. Along with this effect, linagliptin maintains efficacy in patients with CKD while demonstrating a tolerable side effect profile (7).
ADVANCES IN PRACTICE: CKD AND DIABETES MEDICATIONS

GLP-1 Agonists

The GLP-1 agonists have several mechanisms of action including stimulation of glucose dependent insulin secretion, suppression of glucagon secretion resulting in decreased hepatic production of glucose, induction of slow gastric emptying, and promotion of satiety (8).

Exenatide (Byetta®) and exenatide XR (Bydureon®) use in patients with acute kidney injury (AKI) has been studied. While the risk of AKI may be low, it is noted that exenatide is eliminated by renal mechanisms with an increased half-life observed with subsequent decreases in renal function. Thus, exenatide should not be given to patients with severe renal impairment or ESRD (CrCl < 30mL/min) (9).

Data on liraglutide (Victoza®) use in renal impaired patients is somewhat limited. Although liraglutide is not eliminated renally or heptatically, some caution needs to be taken due to this limited data. A meta-analysis of the six Liraglutide Effect and Action in Diabetes (LEAD) studies was completed. Patients received liraglutide 1.2 or 1.8mg daily as monotherapy or in combination with oral antidiabetic drugs or received a placebo for 26 weeks. Patients were separated based on renal function and the results showed that liraglutide was fairly well tolerated aside from increased side effects of nausea that were observed in those with moderate or severe renal impairment (CrCl <60 mL/min). Overall, there was no significant change in efficacy or safety in patients with mild renal impairment (CrCl 60-89mL/min) (9).

The efficacy, safety, and pharmokinetics of a third GLP-1 agonist, albiglutide (Tanzeum) has been studied in patients whose renal function ranges from normal to severe renal impairment. Patients received albiglutide 30mg to 50mg once weekly. Overall, a favorable risk to benefit ratio was seen in patients using albiglutide with varying degrees of renal impairment concluding no recommendations for a dose adjustment of albiglutide in CKD patients (10). Because data on patients with more severe renal impairment is limited, caution should be taken with use in these patients (5).

The utility of dulaglutide (Trulicity®) in patients with CKD is similar to that of albiglutide. Four phase 2 and five phase 3 randomized controlled trials involving a total of 50 patients with mild renal impairment (estimated glomerular filtration rate [eGFR] 60-89 mL/min/1.73m²) and 171 patients with moderate renal impairment (eGFR 30-59 mL/min/1.73m²) treated with dulaglutide were monitored for safety and efficacy. No differences were observed between groups compared to the patients with normal renal function. Due to the limited data in patients with severe renal impairment and ESRD, caution is warranted if dulaglutide is used in this population (11).

Insulin

Exogenous insulin contains only the active form of insulin and is degraded via the muscle, liver, and kidney. About 15 to 20% of insulin metabolism occurs in the kidney (8).

As noted previously, monitoring of blood glucose should be maintained in those with CKD while on insulin therapy. Declining kidney function is associated with an increased risk of hypoglycemia which underlines the importance of blood glucose monitoring. Due to metabolism by the kidney, reduced doses of insulin are recommended for those with renal impairment. This dose adjustment should be individualized for each patient (12).

Meglitinides

The two meglitinides, repaglinide (Prandil® and netaglinide (Starlix®), stimulate insulin secretion from the pancreas. Repaglinide does not rely heavily on kidney function for excretion, and netaglinide is hepatically metabolized with renal excretion of active metabolites (12).

Nateglinide was studied in 40 patients, 20 of which were healthy and 20 who had varying degrees of renal impairment, including ESRD undergoing hemodialysis. Although the amount of nateglinide excreted in 24 hours was lower in the group with renal impairment, the total systemic exposure had no correlation with the level of renal impairment. The drug’s short half-life and short duration of action contributes to the conclusion that accumulation should not be seen and therefore, dose adjustment is likely unnecessary in those with CKD as well as those undergoing dialysis (13).

In another multinational, open-label study, repaglinide safety and efficacy was compared in 151 patients with normal renal function to 130 patients with various degrees of renal impairment. Repaglinide was shown to be tolerable in both groups, with a maximum dose of 4mg three times daily. Final dose tended to be lower in patients with more severe renal impairment compared to those with less severe impairment or normal renal function (P = 0.032) (14). In conclusion, dose reduction in those with more severe renal impairment may be needed but should be tailored to the specific patient (5).

Biguanides

Metformin, an agent responsible for suppression of glucagon, is one of the most efficacious antidiabetic agents. It is eliminated primarily unchanged by the kidneys with little plasma protein binding (15).

Due to the rare but serious risk of lactic acidosis, metformin has dosing limitations for individuals with serum creatinine levels (Cr) of at least 1.4 and 1.5 mg/dL for women and men, respectively. Cr levels below these cutpoints are demonstrative of adequate renal function required to remove 3g of metformin at steady-state levels within 24 to 48 hours (15). There is some debate as to how stringently these recommendations should be followed. The American Diabetes Association (ADA) and European Association for the Study of Diabetes (EASD) reports that metformin use is safe if eGFR exceeds 30mL/min/1.73m² (15). The ADA also suggests that the use of metformin in patients with an eGFR < 60mL/min/1.73m² can be initiated or continued if kidney function is monitored frequently (5).

A pharmacokinetic study confirms the need to dose adjust based on their findings after giving 850mg daily to those with varying levels of renal impairment. Average renal clearance was decreased in mild CKD and mean renal clearance was lower in those with moderate and severe CKD. Maximum concentrations and the area under the curve (AUC) were also increased in...
individuals with moderate to severe CKD compared to those with mild CKD or normal renal function (15).

SGLT-2 Inhibitors

Sodium glucose co-transporter 2 (SGLT-2) inhibitors act by lowering the renal threshold for glucose reabsorption while also increasing glucose excretion. This results in a decreased plasma glucose, osmotic diuresis, and a caloric reduction that promotes weight loss (16). Five randomized, double blind, placebo-controlled, phase 3 trials compared canagliflozin (Invokana®) 100mg and 300mg to placebo in patients with stage 3 CKD. A statistically significant decrease in A1c was noted in patients with decreased renal function, and recommendations of a decreased dose, adjusted according to degrees of CKD. Manufacturer recommendations include reducing the dose from 300mg to 100mg if eGFR is between 45 and 59mL/min/1.73m² and to avoid use if eGFR < 45mL/min/1.73m² (17).

Dapagliflozin (Farxiga®), a second SGLT-2 inhibitor, does not show these same effects in patients with reduced eGFR. Dapagliflozin forms an inactive metabolite called dapagliflozin-3-O-glucuronide (D3OG). Increased amounts of D3OG occur in individuals with declining kidney function. In addition, steady-state renal glucose clearance was reduced by 42 to 84% in mild, moderate, and severe impairment. This higher systemic exposure and decreased efficacy in individuals with renal impairment cause more stringent guidelines on who can receive dapagliflozin (18). Manufacturer’s recommendations include avoiding use in patients with eGFR < 60mL/min/1.73m² (5).

Few studies have been completed for a third SGLT-2 inhibitor, empagliflozin (Jardiance®), in CKD; however, one 52-week randomized, double-blind, parallel-group, placebo-controlled study looked at the efficacy and safety of empagliflozin as an add-on therapy, compared to placebo, in patients with type 2 diabetes and stage 2 or stage 3 CKD. A statistically significant drop in A1c was observed in all groups taking empagliflozin. The adverse effect profile compared to placebo showed that this agent is also well tolerated at all levels of renal dysfunction (19). Manufacturer’s recommendations do not specify dose decreases with declining renal function but do suggest avoiding use in patients with eGFR less than 45mL/min/1.73m² (5).

Sulfonylureas

Sulfonylureas promote insulin secretion from the pancreas. All new generation sulfonylureas, glipizide, glimepiride, and glyburide are metabolized by the liver but only glyburide produces clinically active metabolites. Glipizide does not have active metabolites (8) and glimepiride produces two metabolites, with one being inactive (M2) and the other having 33% of the activity of the parent drug (M1) (5,8).

Of the second generation sulfonylureas, glipizide is the agent of choice in patients with CKD according to the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI) guidelines (20). The lack of active metabolites and the shorter half-life of glipizide (2-4 hours) compared to glimepiride (5-9 hours) and glyburide (10 hours) also contribute to it having a reduced risk of hypoglycemia in patients with CKD. The manufacturer recommends conservative initial and maintenance glipizide doses along with continued blood glucose monitoring (5).

<table>
<thead>
<tr>
<th>Agent</th>
<th>CrCl mL/min</th>
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<tbody>
<tr>
<td>Linagliptin</td>
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<tr>
<td>Sitagliptin</td>
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<tr>
<td>Saxagliptin</td>
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<tr>
<td>Alogliptin</td>
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<tr>
<td>Exenatide</td>
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<tr>
<td>Liraglutide</td>
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<tr>
<td>Dulaglutide</td>
<td>✔</td>
</tr>
<tr>
<td>Albiglutide</td>
<td>✔</td>
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<tr>
<td>Insulin</td>
<td>✔</td>
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<tr>
<td>Repaglinide</td>
<td>✔</td>
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<tr>
<td>Nateglinide</td>
<td>✔</td>
</tr>
<tr>
<td>Metformin</td>
<td>✔</td>
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<tr>
<td>Glipizide</td>
<td>✔</td>
</tr>
<tr>
<td>Glimepiride</td>
<td>✔</td>
</tr>
<tr>
<td>Glyburide</td>
<td>✔</td>
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<tr>
<td>Canagliflozin*</td>
<td>✔</td>
</tr>
<tr>
<td>Dapagliflozin*</td>
<td>✔</td>
</tr>
<tr>
<td>Empagliflozin*</td>
<td>✔</td>
</tr>
<tr>
<td>Pioglitazone</td>
<td>✔</td>
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<tr>
<td>Rosiglitazone</td>
<td>✔</td>
</tr>
</tbody>
</table>

✔ The drug can be given at this CrCl
× The drug is not recommended with this CrCl or no data is available
○ Dose reduction recommended

a 50mg PO once daily (5Cr levels (mg/dL): Men: >1.7 - ≤3.0; Women: >1.5 - ≤2.5)
b 25mg PO once daily (5Cr levels (mg/dL): Men: >3.0; Women: >2.5; or on dialysis)
c 2.5mg PO once daily
d 12.5mg PO once daily
e 6.25mg PO once daily
f limited clinical experience in patients with severe renal impairment or ESRD
g serum creatinine determines if metformin can be given or not
h SGLT-2 inhibitors measured as eGFR (not CrCl)
j do not exceed 100mg/day PO
Decreased levels of glimepiride were observed in patients with moderate to severe renal impairment (5). Those with severe renal impairment conversely had increased levels of the M1 and M2 metabolites by 2.3 and 8.6 times, respectively. This does not restrict the use of glimepiride in renally impaired patients, but suggests dose reduction need to be adjusted based on the degree of renal impairment (5).

Due to the active metabolites and long half-life of glyburide, caution in patients with renal impairment is warranted. The potential accumulation of drug and active metabolites increases the risk for hypoglycemia, therefore, the other sulfonylureas are considered a better alternative (8).

Thiazolidinediones

The mechanisms of action of thiazolidinediones are to enhance tissue sensitivity to insulin and decrease glucose production from the liver (12).

A phase-1 study concluded that the intrinsic metabolic clearance mechanisms of pioglitazone (Actos®) are unchanged in individuals with varying degrees of renal impairment. Also, after repeated 45 mg doses, the mean serum concentration of pioglitazone and its metabolites did not increase, confirming that renal impairment does not cause accumulation and therefore, pioglitazone does not need dosage adjustment (21).

Patients with CKD have a statistically significant increased risk of all-cause mortality, myocardial infarction, stroke, acute coronary syndrome, coronary/carotid arterial intervention, leg revascularization, and amputation in addition to the cardiovascular disease (CVD) risk associated with diabetes. Although patients treated with pioglitazone were less likely to reach these CVD primary endpoints, pioglitazone has been shown to decrease eGFR in those already having CKD as well as increase risk of developing CKD compared to placebo. This places some limitation on the use of pioglitazone in CKD patients (22).

Rosiglitazone (Avandia®) is not considered the thiazolidinedione of choice due to its increased risk of cardiovascular outcomes. Pioglitazone is generally prescribed if a patient is to receive a thiazolidinedione (8). Of note, the issue of volume retention can limit use of this class of medication in patients with CKD and concurrent heart failure.

Clinical Application

The degree of renal function in patients with DM plays a major consideration in selecting appropriate antidiabetic agents. Preferably, agents that rely minimally on the kidneys for metabolism and elimination should be selected for patients who have renal impairment. In addition, decreases in dosage are often recommended in patients with renal dysfunction (Table 1). Linagliptin is one such agent that does not require dosage adjustments and has evidence demonstrating a possible improvement in albuminuria (1).

Sulfonylureas exhibit substantial efficacy if used in a patient with CKD, due to its short half-life and lack of metabolites. However, glipizide will likely still require a dose adjustment to maintain efficacy and safety (5). Despite the fact that all

<table>
<thead>
<tr>
<th>Medication</th>
<th>Use in Dialysis Patients Acceptable</th>
<th>Use in Dialysis Patients Not Recommended</th>
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<tbody>
<tr>
<td>Linagliptin</td>
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<tr>
<td>Saxagliptin</td>
<td>×&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>Sitagliptin</td>
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<tr>
<td>Alogliptin</td>
<td>×&lt;sup&gt;c&lt;/sup&gt;</td>
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<tr>
<td>Exenatide</td>
<td>×&lt;sup&gt;d&lt;/sup&gt;</td>
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<tr>
<td>Liraglutide</td>
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<td>Dulaglutide</td>
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<td>Albiglutide</td>
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<td>Insulin</td>
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<tr>
<td>Nateglinide</td>
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<td>Repaglinide</td>
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<td>Metformin</td>
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<td>Glipizide</td>
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<td>Glimepiride</td>
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<td>Glyburide</td>
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<td>Canagliflozin</td>
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<td>Dapagliflozin</td>
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<td>Rosiglitazone</td>
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</tbody>
</table>

a administer 2.5mg once daily dose after dialysis  
b administer 25mg once daily without regard to timing of dialysis (package insert)  
c 6.25mg PO once daily  
d use is not recommended in patients with CrCl < 30mL/min. Intermittent and continuous hemodialysis will reduce clearance to 0.9 L/hr compared to normal 9.1 L/hr  
e limited clinical experience in patients with severe renal impairment or ESRD

SGLT-2 drugs rely heavily on renal elimination, canagliflozin appears to be the best option. However, it still should not be initiated in patients with CrCl less than 45mL/min. Numerous studies involving canagliflozin demonstrate its utility in patients with renal impairment with only minor dosage adjustments (17). Nateglinide does not require dose adjustment but is a less commonly used agent to treat diabetes due to the inconvenience of frequent dosing along with a risk of weight gain and hypoglycemia (13).

Within the GLP-1 agonist class, although liraglutide is not eliminated renally and has demonstrated efficacy, data is limited in those with severe renal impairment. Therefore, cautious use is recommended (9). Insulin can be used for all levels of CKD but strict monitoring of blood glucose should be employed as well as an individualized dosing regimen. Often, lower doses of insulin are required to achieve glycemic goals (12).
Because there is a statistically significant increased risk of cardiovascular outcomes in patients with CKD, there is concern with use of pioglitazone in patients with the comorbid disease. Additionally, risk of worsening fluid retention and bone loss plagues this class of medications, especially among patients with CKD. If pioglitazone is chosen for a patient with CKD, patient dosages do not need to be adjusted; however, eGFR should be monitored carefully to avoid a decline.

Insulin is recommended for patients undergoing hemodialysis rather than oral agents. This is in part due to the lack of data with oral agents in dialysis. Table 2 outlines which agents have been studied and demonstrated safety and efficacy when used in patients undergoing dialysis. The preferred oral agents are glipizide and repaglinide. The hepatic metabolism of these agents results in lower risk of hypoglycemia and makes them appropriate choices in those with CKD (12).

Ongoing glycemic monitoring is also a crucial aspect in CKD patients taking diabetes medications. HbA1c monitoring is recommended at baseline and every 3 to 6 months with a goal of less than 7.0% (23,24). An annual urinary albumin-to-creatinine ratio is also recommended as well as baseline and periodic serum creatinine (23). The NKF KDOQI guidelines use eGFR to stage the level of CKD; therefore, it should be monitored periodically to determine the current stage of the patient and adjust medication doses appropriately (24). Due to the potential for increased risk of hypoglycemia with some agents and the possibility of appetite changes with ongoing CKD, self-monitoring of blood glucose is essential in these patients. Such monitoring also aids in prevention of further kidney injury and microvascular complications.

Summary

The high prevalence of patients with DM and CKD reaffirms the necessity of ensuring patients are on correct antidiabetic agents. Selecting the most appropriate pharmacological agents in these patients may reduce the risk of microvascular complications, prevent further kidney impairment, and improve quality of life. Frequent monitoring will help in adjusting therapy as needed to provide the best regimen for the patient. Through careful selection and continual monitoring, an individualized care plan can be created for these patients that maintains safety and efficacy.

References


PRACTICING MINDFULNESS IN DIABETES SELF-CARE

Mark Heyman, PhD, CDE
Solana Beach, CA

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Living with diabetes can be overwhelming, and many of your patients may experience strong emotions related to their condition. When faced with difficult diabetes-related emotions, it’s normal to spend a lot of time and energy trying to feel better. One common strategy is to work to avoid such unpleasant feelings. Avoidance can come in many shapes and sizes. Some people watch mindless television, while others sleep, exercise, or binge eat or drink. Often people with diabetes avoid actively managing their diabetes because doing so may bring up thoughts about themselves and feelings that they want to avoid.

The question that we need to ask our patients is how well do these avoidance strategies work in achieving their goals? Although some people find that avoidance helps them in the short term, such approaches do not eliminate negative feelings in the long term. In addition, avoidance strategies may exact a high cost. For example, spending a lot of time sleeping to avoid thinking about overwhelming feelings about diabetes leaves less time to spend with friends and family and stay healthy by managing diabetes.

What if you asked your patients to try not to avoid their uncomfortable diabetes-related emotions but instead simply to observe them? What if you told them that doing this may help them focus less on their negative feelings and allow them more freedom in their lives? What if you asked your patients to be mindful?

What is Mindfulness?

Mindfulness means paying attention on purpose, in the present moment, and nonjudgmentally, to what you are experiencing. In other words, observing the thoughts, emotions, and physical feelings you are having right now without avoiding or judging them, rather simply noticing them. The goal of mindfulness is not to make one feel a certain way (e.g., relaxed or happy). The goal is to give the person space to simply notice the present experience. Research shows that mindfulness can be used to reduce stress and symptoms of depression and anxiety as well as improve cognitive flexibility and social relationships, all of which support effective diabetes management.

Being mindful can be hard, and being mindful when living with diabetes can be even more difficult. Diabetes management is overwhelming, and when a person is busy doing everything necessary to manage his or her diabetes, paying attention to experiences in the present moment can be especially difficult. An even greater challenge is to pay attention to experiences without judgment; most people with diabetes beat themselves up, at least sometimes, when their blood glucose is out of range. Registered dietitian nutritionists can teach mindfulness to their patients to help them take a different approach to managing their difficult diabetes-related emotions.

How can your patients benefit from taking a more mindful approach to their diabetes management? As importantly, what is the alternative? Practicing mindfulness can help patients learn something new about themselves. They may see that even though it is not always easy, they can handle the uncomfortable emotions they feel about diabetes. They may even find that experiencing these uncomfortable emotions is not as bad as they imagined. The feelings may still be uncomfortable, but practicing mindfulness can blunt the ability of difficult emotions to control behavior. Mindfulness allows people to choose their behavior, rather than letting their emotions dictate how they behave. In contrast, not being mindful or working to avoid uncomfortable thoughts and emotions requires the investment of substantial time and energy with no real benefit; most people cannot banish negative thoughts and emotions on demand.

How Can Patients Practice Mindfulness?

How can you help your patients practice mindfulness in their lives and in their diabetes management? Following are several specific techniques:

Mindful eating: Ask your patients to pay attention to their experience as they are eating: As you eat, notice any thoughts you have about your food. Are you looking forward to eating it? Are you feeling guilty about eating it? Are you worried about what the food will do to your blood sugar? Take some time and notice the different sensations you experience as you eat. How does the food feel on your lips, your tongue, as you swallow? What does your food taste like? What is the temperature? The texture? How spicy is it? Just notice, without judgment, what you experience as you eat your meal. Try eating one meal mindfully and see what you notice and learn.

Mindful glucose monitoring: Ask your patients to pay attention to their experience while checking their blood glucose: Before you check your blood sugar, notice your thoughts. Do you think your blood sugar is high, low, or in range? How does this make you feel? Content? Discouraged? Notice what it feels like to prick your finger. Notice all the steps involved in putting the drop of blood on the strip. When you see the result, notice your response. Are you surprised? Proud? Frustrated? Once you have the results, take a minute to observe what you just experienced, not from a place of judgment but from one of curiosity.

It’s important to remember (and to remind your patients) not to get discouraged. Mindfulness requires ongoing practice. Also remind your patients that the goal is not to make their experiences go away but to notice them, both pleasant and unpleasant experiences. Mindfulness can give them the space to observe their emotions without letting the emotions control them. This space can help them make decisions based on their real desires, rather than what their emotions are “telling” them to do.

Finally, and perhaps most importantly, it is important to understand that it is not possible to teach others to be mindful unless you practice mindfulness yourself. Only through experiencing mindfulness can a person gain a true understanding of the process. Practicing mindfulness can help clinicians manage stress in their work and allow them to effectively teach patients about the benefits of the practice in diabetes management.

Jon Kabat-Zinn sums up mindfulness nicely with this quote:
“You can’t stop the waves, but you can learn to surf.”

Mark Heyman is a trained clinical psychologist and certified diabetes educator who has lived with type I diabetes for over 15 years. He is the founder and director of the Center for Diabetes Mental Health. To learn more about Mark and his mission to provide evidence-based mental health services for people living with diabetes as well as training for health care professionals, visit his website: www.cdmh.org and follow him on twitter @DiabeticPsych.
Insulin Pump Therapy and the Dialysis Patient

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I worked several years as a renal dietitian and never saw a patient with an insulin pump even though about sixty percent of my patients had diabetes. Once I began working as a Diabetes Clinical Manager with an insulin pump company, I soon realized how little I knew about insulin pumps and their amazing ability to help patients achieve tighter control of their blood sugars. Insulin pumps use rapid-acting insulin which deliver insulin as basal and bolus. The basal insulin covers the body’s psychological non-food needs for insulin while the bolus is used to cover carbohydrate intake and corrections of high blood glucose.

Insulin pumps reduce the burden of diabetes by assisting patients in determining their insulin dose based on their carbohydrate ratio, insulin sensitivity, active insulin and their blood glucose target range. Also, because of the pump’s delivery mechanism and absorption, total daily insulin dose is typically reduced by twenty-five percent. The inherit variability of injectable insulin is reduced from 30% to less than 3%. Insulin is placed into the pump and is delivered to the body via tubing/infusion set. The infusion set is changed every 2-3 days and there are multiple types of infusion sets to meets a patient’s preference or needs. Pump settings can also be customized to allow insulin delivery based on an individual’s cognitive capacity.

Several manufacturers offer insulin pumps for insulin delivery therapy, but look for a pump that will suspend delivery of basal insulin for up to two hours if the sensor glucose hits a preset low and the patient doesn’t respond or chooses to allow the pump to suspend. These pumps will then resume basal delivery for four hours and if the sensor blood glucose is still low it will suspend for an additional two hours. The benefit of this feature is reducing the impact of hypoglycemia on patients especially for those individuals with hypoglycemia unawareness. Since the majority of severe hypoglycemia occurs at night, there is an added peace of mind when an individual goes to bed knowing that the pump will take action for them when they cannot. A sensor is part of a Continuous Glucose Monitoring System (CGM) which is worn on the body for up to six days. The system measures interstitial glucose every five minutes, or 288 sensor glucose readings per 24 hours. Patients are able to keep constant track of their glucose levels which gives them a complete view of how medication, food, stress and exercise affect their blood glucose. New pumps coming on the market will adjust the basal insulin delivery as blood sugar approaches either high or low.

I have recently trained three dialysis patients and each commented that they wish they had been started on an insulin pump earlier. They have experienced improvement in their A1Cs and each stated that they were feeling better with increased energy levels. Insulin pumps offer a clinical and lifestyle alternative to standard insulin therapy in controlling diabetes yet the majority of dialysis patients are not offered pump therapy. Erroneous belief that the pump is too costly or too difficult to use is unfounded. Better glucose control means less hospital admissions which results in less missed renal replacement therapy days. Also, insulin used for a pump is billed under Medicare Part B rather than Part D which may reduce the cost of insulin contributing to the prescription “donut hole”.

Renal dietitians are responsible for clinical outcomes such as albumin and fluid control. Pump therapy may assist dialysis patients in achieving improvement in these outcomes. Also, pump therapy allows individuals greater flexibility in their eating patterns and variable insulin needs of dialysis and non-dialysis days. I have found that an insulin pump is a tool that can positively impact a patient’s life by reducing the burden of diabetes therapy and allowing patients greater freedom and better health. As you pass out this month’s nutrition report card, consider which one of your patients may benefit from insulin pump therapy.
RPG RESEARCH

Ratio of Hemodialysis (HD) Patients Per Allied Health Professional
Renal Practice Group Funded Research Presented at NKF Spring Clinical Meetings
Rosa K Hand, MS, RDN, LD, FAND
Director, Dietetics Practice Based Research Network at the Academy of Nutrition and Dietetics

As Director of the Dietetics Practice Based Research Network (DPBRN) at the Academy, I am always looking for important research questions and for DPG partners who can help answer them. When I was in my Master’s degree program, I conducted a survey of renal dietitians (RDNs and international dietitians) exploring their ability to conduct diet assessment (1). Along the way, we discovered that 25% of RDNs reported having more than 150 patients per full time equivalent (FTE), the maximum ratio recommended by the KDOQI Nutrition Guidelines (1). As a result of that study, several prominent physicians wrote a commentary in the Journal of Renal Nutrition stating their belief that bone and mineral disease protocols were taking up too much RDN time and detracting from their ability to conduct diet assessments (2). This surprised me and my co-investigators, so we conducted a DPBRN survey that asked RDNs to rank their job responsibilities in terms of their importance and the amount of time consumed. In that study, half as many (11.9%) of RDNs were responsible for more than 150 patients/FTE (3). We discovered that plan of care related activities were considered both highly important and highly time consuming, and learned about new roles for RDNs related to pharmacy benefits management (3).

Simultaneously, I was working with the Clinical Nutrition Management DPG to conduct a study on staffing levels for inpatient RDNs. This study, conducted in 2014, was able to demonstrate how many patients RDNs were responsible for, but not whether more RDN time was related to better patient outcomes (4). Because dialysis RDNs are responsible for patients over long periods of time and since nutrition is such an important part of their outcomes, I began to wonder whether we might combine these two ideas and measure the impact of RDN staffing levels in dialysis facilities and the relationship to patient outcomes. I proposed this project to RPG and they agreed to provide funding so that the DPBRN could conduct the project. Working with an advisory group from RPG (Mary Kay Hensley, Cathy Goeddeke-Merrickel, Jessie Pavlinac, Jerrilynn Burrowes, Lesley McPhatter), a research nephrologist who is a great advocate for nutrition and RDNs (Ashwini Sehgal, MD) and a statistician (Jeffrey Albert, PhD), we developed methodology that would determine whether fewer patients per RDN benefited dialysis patient outcomes. Because gathering outcomes on a per patient level would be very time-consuming, and so much data is already gathered, we decided to approach this question with data from the CMS Annual Facility Survey (AFS), on which dialysis facilities have to report the number of full and part time RDNs, social workers, nurses, and patient technicians each year. This data is matched with facility standardized outcomes as well as number of patients who receive care at the facility. From this information, we estimated a patient:FTE ratio for each type of staff member. Other researchers have estimated staffing ratios from the AFS data in the past to describe what facility characteristics are associated with variations in staffing levels (5).

The poster presented at NKF and republished here shows the trends for patient:staff ratios over the four years of data we had available. It appears that the ratios are decreasing, which should be good news, but we don’t know whether this is accompanied by an increase in responsibilities or other factors. In addition, the figure shows that the estimations of ratios may be too low, because of the non-specific way in which part and full time positions are reported on the AFS. The next step is to associate these ratios with patient outcomes. We are working on this analysis now, and hope to be able to present a portion of it as a poster at FNCE 2017.

In addition, we are using direct observation methodology to quantify the time renal RDNs spend with patients vs on indirect care activities because we think this may provide useful information, particularly if higher ratios appear to benefit patient outcomes. That might indicate that RDNs with higher ratios are spending more time in direct care vs. in other activities.

As I discussed with some RPG members who visited the poster at NKF (see photo), this question about renal staffing ratios was brought into the spotlight with the California legislature’s interest in mandating ratios for patients per nurse and social worker but not RDN. The data from this project should help make evidence-based staffing recommendations and legislation in the future, so thanks to RPG for providing funding for this project.

The poster will also be available at RPG’s website, www.renalnutrition.org.

References
Background

• Nurses, patient care technicians (PCTs), registered dietitian nutritionists (RDNs) and social workers (LSWs) are required members of the interdisciplinary team for dialysis.
• The mandate from the Centers for Medicare and Medicaid Services (CMS) does not specify patient: staff ratios.
• Some states do mandate maximum ratios.
• The KDOQI Nutrition Guideline specifies that full time equivalent (FTE) RDNs should not be responsible for more than 150 patients.
• Average ratios of 120 patients per FTE RDN have been reported in survey data in the past (2, 3).

Methods

• Data were from the 2009-2012 CMS Annual Facility Surveys (AFS).
• Data were purchased from ProPublica, which obtained the data through a Freedom of Information Act Request.
• We included facilities in which all 4 years of data showed at least 30 hemodialysis (HD) patients and 0 pediatric patients (n=3891).
• Staffing is reported for the 4 professions on the AFS as full time (>32 hours per week) and part time (PT) positions.
• We used formulas (below) to estimate patient: professional ratios based on the AFS reports.
• We calculated mean and standard deviation for the ratios each year, and the change over time using repeated measures ANOVA.
• We also calculated the number of facilities with >120 or >150 patients per RDN and LSW.

Results

• Patient per FTE professional ratios decreased over the 4 years of data (p<0.001 for all by repeated measures ANOVA) (Table 1).
• The percent of facilities with patient: RDN or LSW ratios >120 or 150 declined (Fig 1).
• The low precision of the AFS reports may lead to over- or under-estimation of ratios compared to estimation based on hours (Fig 2).

Table 1: Mean ratios of patients per allied health professional in 4 years of AFS data

<table>
<thead>
<tr>
<th>Patients per:</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nurse</td>
<td>17.4±9.0</td>
<td>17.2±8.8</td>
<td>16.2±7.5</td>
<td>16.0±7.4</td>
</tr>
<tr>
<td>Nurse per shift</td>
<td>4.8±2.9</td>
<td>4.6±7.8</td>
<td>4.3±2.3</td>
<td>4.3±2.3</td>
</tr>
<tr>
<td>PCT</td>
<td>12.1±6.2</td>
<td>11.9±4.5</td>
<td>11.6±4.9</td>
<td>11.7±4.4</td>
</tr>
<tr>
<td>PCT per shift</td>
<td>3.4±2.2</td>
<td>3.3±1.9</td>
<td>3.2±1.7</td>
<td>3.2±1.9</td>
</tr>
<tr>
<td>RDN</td>
<td>98.7±37.7</td>
<td>99.3±37.1</td>
<td>94.3±35.2</td>
<td>92.4±34.2</td>
</tr>
<tr>
<td>LSW</td>
<td>94.7±34.5</td>
<td>95.2±35.0</td>
<td>91.9±33.4</td>
<td>90.5±32.8</td>
</tr>
</tbody>
</table>

Conclusion/Discussion

• HD patient: staff ratios have decreased over 4 years for PCTs, nurses, RDNs, and LSWs.
• While statistically significant, the clinical significance is unclear.
• The ratios calculated from AFS data are lower than those reported by RDNs in survey data.
• This is likely due to the fact that a PT position reported on the AFS could represent anywhere from 1-31 hours per week.
• We included facilities in which all 4 years of data showed at least 30 hemodialysis (HD) patients and 0 pediatric patients (n=3891).
• Staffing is reported for the 4 professions on the AFS as full time (>32 hours per week) and part time (PT) positions.
• We used formulas (below) to estimate patient: professional ratios based on the AFS reports.
• We calculated mean and standard deviation for the ratios each year, and the change over time using repeated measures ANOVA.
• We also calculated the number of facilities with >120 or >150 patients per RDN and LSW.

References:


A copy of this poster will be available to download on the website www.renalnutrition.org in the Newsletter Archives.
RPG CHAIR MESSAGE

Annamarie Rodriguez, RDN, LD
RPG Chair

As spring approaches and we focus on transition it is a time for not only change and a new beginning, but of retrospect and identifying a course of action for the coming year. At our recent Spring Transition meeting we spent time reviewing our perception of RPG’s (Renal Practice Group) Strengths, Weaknesses, Opportunities and Threats using a SWOT analysis and, at the end of our meeting, compared it to the previous years’ SWOT analysis. We found while we still have a journey ahead of us, we had actually met an amazing number of goals and continue to remain a constant, positive, vibrant, professional resourceful organization of which I am proud to start my term as Chair.

Some of our strengths included collaboration, networking, continuing education, RNF (Forum), educational handouts, scholarship opportunities, webinars to study for the CSR exam, lending library… and more.

Some of our weaknesses included minimal diversity, limited communication and limited visibility. The list was longer but I’d like to focus on the positive. However we did chuckle amongst ourselves when we noted that communication was listed only once this time, however listed 3 times last year. We are rising to the task with this ongoing goal.

Our committees are growing and we’d love to share these opportunities with renal dietitians who may be interested. Growth is one of the challenges I am pursuing with our team. Grow our committees, ‘build the ballots’, and grow membership, interest, and involvement. We have many resources to offer and one of the challenges we face is getting the ‘word out’. Be on the lookout for announcements in the Forum, eBlast and now through Social Media – which is another growing opportunity.

As you might be aware this is a milestone year for the Academy; if you have never attended FNCE in the past I urge you to attend this year. Attend the Food & Nutrition Conference & Expo October 21-24 in Chicago and celebrate the Academy’s “Centennial”. In addition, join us during FNCE for the RPG’s Spotlight Session. Dr. Kam Kalantar-Zadeh and Kristin Leonberg, MS, RD, CSR, LD/N will present “Approach to Malnutrition in Progressive Chronic Kidney Disease” with a deep dive into the complex nutritional management of CKD with a discussion on protein energy wasting and the impact that dialysis has on the complex paradigm of protein energy malnutrition and inflammation. The session utilizes evidence based practice with Medical Nutrition Therapy – something for all renal RDs.

I’m already looking forward to my next message. I’d like to share more information about our progress and benefits. I’d also like to hear from you, the RPG members. We welcome feedback. I’d like to know what your perception of RPG is.

Like and follow our Facebook (Renal Dietitians – RPG) page and Twitter account (@RenalRDNs) and… SHARE with your colleagues. Until next time… #keepinitrenal

CRN CHAIR MESSAGE

Laura Holden, MBA, RD, CSR FNKF
NKF/CRN Chair

This issue marks my first message as NKF/CRN Chairperson. Previously I have served on the CRN Executive Committee in various positions for the past 10 years. Along the way, I served with and met, many wonderful renal dietitians, and am indebted to them for all their wisdom and advice.

As I write this, the National Kidney Foundation is preparing for their annual Spring Clinical Meeting, which this year is in Orlando, Florida, April 18-22. During this meeting, the CRN Executive Committee will have had the honor of meeting with RPG’s Chair, Annamarie Rodriguez to discuss CRN/ RPG joint projects. One area under discussion will be the next revision of our joint publication: A Clinical Guide to Nutrition Care in Kidney Disease. We are looking for interested Academy/RPG and NKF/CRN members who are willing to volunteer some time to help with this joint project. We are starting to form our committee now in anticipation of the new KDOQI Guidelines anticipated to be released sometime in 2018. I welcome anyone interested in assisting with this project to contact either myself or Annamarie Rodriguez. CRN and RPG are continuing their collaboration on the National Renal Diet. This too awaits final revisions until after the 2018 update to the KDOQI Guidelines.

I am also pleased to announce CRN’s most prestigious award, the Joel D. Kopple Award, was presented to Tilakavati Karupaiyah, PhD, APD, an Accredited Practicing Dietitian (APD) with the Dietitians’ Association of Australia, and Associate Professor and Head of the Dietetics Program at the National University of Malaysia. She is an adjunct associate professor at Wayne State University in Detroit. Dr. Karupaiyah became involved in renal nutrition because of a lack of dietitians in the field in Malaysia. She is now working to build mentorship programs for developing renal dietitians in Malaysia through engagement in nutrition research.

CRN’s Recognized Renal Dietitian this year is Lesley McPhatter, MS, RD, CSR, from the University of Virginia Health System in Lynchburg, Virginia. Lesley manages 13 renal dietitians and precepts 12 dietetic interns each year. She is involved with CDR as an item writer for the Certified Specialist in Renal Nutrition exam and has been an active member of her local CRN chapter.

This year CRN also awarded a 2 year, $70,000 research grant to Abigail Eldridge, RD of the Colorado’s Children Hospital to help develop renal nutrition guidelines for children from birth to 2 years old. I am excited to read the outcome of Abby’s research in the future and hope it will be beneficial to our youngest CKD patients.

I would also like to take this opportunity to remind everyone the NKF continues to offer free CEUs to members through its Professional Education Resource Center (PERC). These CEUs are offered to non-members for a fee. I have had renal dietitians in the past tell me they have used these CEU opportunities to help them study for the CSR exam and meet their CDR goals.

Please feel free to contact me with any questions you have regarding CRN, if there is a project your are interested in working on, or have a suggestion for a future project or area of concern for CRN. I am looking forward to hearing from you.
7 Day Food Journal Challenge

Christine Farinella
Dietetic Intern, University of Delaware
Newark, DE
Email: iscmf@udel.edu

Many apps have been developed to help consumers with their food choices and food tracking to live a healthier and more mindful life. One of these apps, the 7 Day Food Journal Challenge, allows users to track their foods by way of a visual food diary; you take a picture of your meal and the app will log it for you. The app goes beyond tracking food; it allows you to follow topics that interest you such as healthy eating, gluten free living, paleo diets, vegan diets, weight loss, etc. Following boards connects the user with other members of the community who share the same interests and topics as you; the purpose is to create a support group and share inspirations with other members. Deciding to make your posts public can help you connect with different members, but you can also keep your photos private and still be involved with the interest groups.

Those who have tried calorie counting and tracking and found it to be stressful and unsuccessful may find this app to be more beneficial. This app allows you to share your meals, creativity, and support to others in an online community. Even if you have a bad day, you still have a diary of past photos that serves as a reminder of past successes, and can inspire you to keep trying. Focusing less on calories and exact portion sizes can be overwhelming, but this app takes the pressure off of exact amounts, and focuses more on overall healthy eating.

Even though this app has some good aspects it also has some significant drawbacks, enough to make a strong recommendation against the app as a resource particularly for renal patients. These drawbacks include a lack of a board for people who are interested in renal diets, it does not provide a nutrient breakdown for logged in foods, and it requires a $2.99 monthly subscription after the end of a 7-day free trial. Although having a visual aid for meals may help remind clients of what they consumed, it does not provide any nutrition information. However, this app can be helpful when conducting a 24 hour recall to find out what has caused a patient’s potassium or phosphorus to spike, but simply taking a picture with your camera and saving it would work just as effectively.

Anyone following a particular diet with restrictions will benefit from the nutrient breakdown of food. Having a resource to fulfill this need is essential for renal patients who need to closely monitor their intake of sodium, potassium, phosphorus and protein. Unfortunately, this app does not provide this information and does not provide the patient with any feedback or guidance. Finally, the fact that this app requires a monthly subscription is off-putting, considering your camera phone can fulfill this purpose just as well. Overall, this app would not be beneficial particularly towards renal patients, but may be beneficial for someone who is motivated and tuned in to social media to showcase their eating habits and be held publicly accountable.

APP REVIEW

FNCE RPG Spotlight

Tuesday, October 24, 2017: 9:45-11:15 AM

At this year’s FNCE RPG Spotlight Session, Dr. Kam Kalantar-Zadeh and Kristin Leonberg, MS, RD, CSR, LD/N will present “Approach to Malnutrition in Progressive Chronic Kidney Disease”.

This session offers a deep dive into the complex nutritional management of people with chronic kidney disease (CKD) with a discussion on protein energy wasting and the impact that dialysis has on the complex paradigm of protein energy malnutrition and inflammation in the CKD population. It provides a review of the impact of the physiological changes of kidney function on nutritional status as CKD progresses. The session offers practical solutions and management of malnutrition and inflammation in CKD by utilizing evidence based practice with Medical Nutrition Therapy.
Looking Forward as We Move Toward Our Next 40 Years

Mary Kay Hensley, MS, RD, CSR
Second Century Liaison

The Academy is charting a new vision for the future, grounded in an extraordinary commitment to collaboration, a focus on service and an emphasis on accelerating the progress towards solving the greatest food and nutrition challenges of the 21st century—creating a world where people and communities thrive through the transformative power of food and nutrition.

I asked both long term members who have practiced nephrology nutrition for many years, some as long as 50 years, and current Executive Committee members the following question:

“Using your crystal ball, what do you see for the future of renal dietetic practice?”

Their comments provide some similarities and contrast. What are your thoughts?

Donna Bodnar, MS, RD, CSR
Cleveland, OH, practicing since 1979
Iron may be the aluminum and calcium of this decade. Renal dietitians need to stay on top of their game. We need to use our diagnostic skills and not just push protein and binders because others can easily replace us in those roles.

Lois Hill, MS, RD, CSR, LD
Lexington, KY, practicing since 1972
My dream would be that the renal dietitian will move to more proactive, preventative nutrition counseling in earlier stages of CKD. Along with this dream, I would hope for an efficient payment system so that RDNs could earn an income that is more financially rewarding than what we are currently earning in the dialysis world. In other words, there would be a requirement that all CKD patients be referred to an RDN.

Judy Beto, PhD, RDN, LD, FAND
Chicago, IL, practicing since 1973
We have to continue to step up to the plate and be recognized for the essential role we play in caring for renal patients. I see many renal dietitians overwhelmed with high patient care loads. We are overwhelmed with paperwork and documentation, but yet that is the only way we can document that what we do makes a difference. There are renal positions open all over the United States. We will never be without a job, but what we have to do is become even more valuable monetarily by demonstrating that our care keeps patients living longer. Why recruit a new patient when we can keep our current ones healthier longer within our units? We have to solve that puzzle and connect the dots to keep our worth high and go even higher!

Jean Stover, RDN, LDN
Philadelphia, PA, practicing since 1973
I only hope that RDNs involved with the care of CKD patients, including those receiving dialysis can keep up with CMS and dialysis corporation requirements while still achieving adequate nutritional status and good biochemical control for their patients.

Theresa (Terrie) Rydzon, RD, LDN, LND
Chicago, IL, practicing since 1976
The challenge to keep up with all the new food products on the market, especially those with food additives containing phosphorus, sodium, calcium etc. will continue to be a concern for RDNs. We have to stay current with our population’s eating habits so that we can make effective renal diet guidelines for our patients. We continue to move towards collaborative care of our patients with other members of our health care teams, which is a bonus for renal RDNs.

Faye Moore, RD, CSR
Phoenix, AZ, practicing in renal since 1988
I see more focus on patient readiness to learn and team collaboration in the future.

Connie Schroepfer, MS, RD
Oakland, CA, practicing since the late 1960s
I am afraid the future practice of renal dietetics is corporate and lab results driven. RDs no longer input the data for kinetics and are not extensively trained to evaluate the data results or potential treatment errors. When units are evaluated using the number of lab results in the desired range, those numbers rather than the whole patient, become the focus. I hope there will be more emphasis on renal RDNs learning more about general medical conditions and staying abreast of basic nutrition science research.

Rita Solomon-Dimmitt, RD, CSR, LDN
Nashville, TN, practicing since 1977
We should all be involved in promoting home dialysis to engage the patient and empower them to be involved in their care...
and maximize their success. CKD has had such advances in the level of care and improvement in technology during my career. I hope that we can continue to offer new and innovative techniques that will improve the lives of patients and make dialysis less intrusive and more effective, allowing them to continue to participate in activities that they enjoy.

Carol Liftman, MS, RD, LDN
Philadelphia, PA, practicing since 1983
I hope renal dietitians will continue to expand their role in the total care of the patient. Allowing and expanding the delegation of authority to dietitians to make medication changes using protocols will improve patient care.

Maureen McCarthy Diamond, MPH, RD, CSR, LD
Portland, OR, practicing since 1986
I am concerned that it will be a struggle to maintain the profile we as renal dietitians have enjoyed so far. Renal nutrition continues to be an exciting specialty, one that requires a knowledge base that is broad and deep. That is the challenge that attracts many of us to this specialty. It is also the challenge that puts continued high profiles for renal dietitians at risk. It takes a lot of work, a lot of team work. In this case the team I’m thinking of is the group of dietitians that have represented us in the Renal Practice Group (RPG) and the Council on Renal Nutrition (CRN). I hope these two groups can continue to attract bright volunteers who can think beyond the envelope to assure future strong roles for renal RDNs.

Judith Heath, MS, RD, LD, FAND
Nominating Committee Chair, practicing since 1988
I foresee more use of motivational interviewing to engage patients rather than police them. I think large dialysis companies will continue to be major players in the renal field with less independently owned centers. I think RPG will need to partner with major employers in this field or become obsolete.

Sandra Daws, RDN, LD
Treasurer, practicing since 1988
I predict more intensive bone mineral metabolism management.

Melissa Prest, MS, RD, CSR, LDN
Electronic Media Manager, practicing since 2008
I think RDNs will become more involved with fluid management, will increase the number of physical assessments performed, will incorporate the nutrition care process into their practice, and that mentorships with advanced practice renal RDNs will become readily available.

Jennifer Parker, RDN, LDN
Patient Education, practicing since 2005
My wishful thinking includes a concierge model that services patients in CKD stages 1-5; less entities that produce the same types of patient education materials; multi-partner private practice institutes or centers for nutrition therapy where MDs or other prescribers consult with us, so that we are the central source of CKD management; and a better Academy, one that creates “nutrition summits”.

Sara Erickson, RD, CSR, LDN
Chair-elect, practicing since 2002
I work in pediatrics and I would like to see increased involvement of RDNs as patients transition to adult centers/practices.

JoAnn Randazzo, MS, RD, CDN
Past Chair, practicing since 2007
I see a future focus on CKD 1-4 education and funding focused on early intervention education, including diet education, to delay the progression of CKD.

Rose Johnson, LRD
Secretary, practicing since 2007
I predict increased involvement for RDNs through the Interdisciplinary Team process.

Annamarie Rodriguez, RDN, LD
Chair, practicing since 1997
I foresee a global, streamlined unified approach between health care providers transcending, not just CKD, but other disease co-morbidities. This approach embraces early management and preventive education of CKD stages 3 and 4 which progresses into renal replacement therapy with available coverage options.

Donna Gjesvold, RDN, LD
Reimbursement Chair, practicing since 1995
I predict no more jobs for renal RDNs once artificial kidneys are cheaply available so patients no longer have to work at healthy eating.

Thank you!

For your continued support of the Foundation and our Second Century! We would like to highlight our members who have generously donated to the Second Century

Campaign:
• Beseler, Lucille
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• Farr, Linda T
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• Lakshmikantham, Rupa
• Martin, Donna S
• McCarthy, Maureen P
• Nagy, Judith A
• Pace, Rory C
• Pavlinac, Jessie M
• Schofield, Marsha K
• Visocan, Barbara J
• Walters, Nancy G

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RECENTLY PUBLISHED

Insufficient time to check out recently published articles in nephrology nutrition? In an effort to help keep our RPG members current, we reviewed the following articles from a variety of publications. We hope you find this list helpful and, as always, would appreciate your feedback and suggestions!


This prospective study of 1,658 dialysis patients evaluated patients’ frailty phenotype and looked at patient outcomes during a median follow-up period of 17 months. The authors found significant associations with frailty and higher rates of both hospitalization and mortality.


The phosphate content of prescribed medications for 101 hemodialysis patients was reviewed in this study. Out of 124 medications, 11% contained phosphate with central nervous system and cardiovascular medications accounting a majority (89%) of the phosphate-containing medications.


This study provided an individualized plan to replace foods with phosphorus additives with foods of similar nutritional value without additives to an intervention group of 67 dialysis patients. The control group received regular care from their renal dietitians. The authors found a significant decline in phosphorus levels in the intervention group after 3 months without compromising nutritional status.


In this retrospective study, 122 hemodialysis patients were assessed for sarcopenic obesity using dual energy x-ray absorptiometry. The researchers looked at multiple definitions for sarcopenic obesity, and found no association with mortality with any of the current sarcopenic obesity definitions.


Fifty-six kidney transplant patients were randomized to receive either 3 g of L-carnitine or a placebo starting the day before kidney transplantation to determine if L-carnitine supplementation could have a protective effect against delayed graft function. No protective effects were found with L-carnitine supplementation.

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**How to Donate to the Second Century Initiative**

- Donate online by selecting the Second Century initiative and be recognized within your appropriate giving level.
- If you prefer to make a pledge over a number of years, please fill out the Second Century pledge form and email to pslomski@eatright.org.
The nutrition focused physical exam gained a lot of interest in recent years since it is a useful tool to help diagnose malnutrition. The “Assessing Nutrition Status Using Nutrition Focused Physical Exam” session was one of the most popular topics and attracted many dietitians at the National Kidney Foundation Spring Clinical Meeting 2017. Laura Olejnik was the presenter who has a background in critical care and currently works at dialysis centers in Chicago. In Laura’s presentation, she focused on head and neck exams as well as dysphagia screening.

There is a difference between a nutrition focused physical assessment (NFPA) and nutrition focused physical exam (NFPE). Many dietitians may already be practicing NFPA which includes biochemical data, anthropometrics and other objective findings from other disciplines. In a NFPE, a dietitian performs the exam by measuring body composition and vital signs. She may also look for signs of nutrient deficiencies. It is important that the patient is alert and oriented before performing a physical exam. Permission must be obtained from the patient. RDs should explain the purpose of the exam and that she is looking for signs of nutrient deficiencies.

To begin the physical exam, first a RD can put her fingers on both sides of the patient’s jaw. Then she may ask the patient to open their jaw as wide as possible. She will observe if the movement is symmetrical and the range of movement. The trigeminal nerve controls motor functions and strength of the temporomandibular joint. A weak jaw may indicate deficiency in the trigeminal nerve. If the range of movement is small, the patient may not be able to tolerate bigger bites. Second, for the facial exam, the patient is asked to fill his or her cheeks with air and try to hold it in. The RD will place two fingers on the cheeks and push on them gently to see if any air escapes. Poor lip seal may be an indicator for facial nerve deficiency and is related to pocketing and drooling. The RD may ask the patient to stick his or her tongue out and observe if the tongue is in the midline. She may also ask the patient to move the tongue in different directions to assess the range of motions and strength. Abnormality in this area is related to a higher risk for aspiration and suboptimal oral intake. For the oral exam, ask the patient to say “ahh” and observe if the movement of the soft palate is symmetrical or the uvula is deviated. An abnormal result may indicate glossopharyngeal and vagus nerves deficiency. Finally, the trapezius muscles on the shoulder may also be evaluated. This muscle helps support the head and neck in an upright position to allow the swallow passageway to be open.

Nutrition focused physical exam may be a valuable tool for RDs to diagnosis or identify malnutrition. Additionally, it may also uncover underlying problems that affect oral intake such as neurological deficit or dysphagia.
Need funding to attend a conference? Interested in research? Looking to pay your tuition?

Awards, Grants, Scholarships and Stipends

RPG offers members a chance to enhance their knowledge through CEU opportunities obtained in the classroom or a meeting setting. Members can find applications and descriptions for financial awards, grants, or scholarships on the RPG website under the Professional and Student Resources tab, followed by Grants and Scholarships. Each application has its own link listed next to each financial award, grant, or stipend. Below is a summary of what is available online.

Conference/Meeting Stipend Award
• RPG members can apply for a conference/meeting stipend award to attend a nephrology or renal related educational seminar or conference of their choice.

Research Grant
• RPG offers research grants for a member conducting an original research project in an area related to or benefitting those with chronic kidney disease.

Scholarship
• RPG offers scholarships for members pursuing a post-baccalaureate degree in a field applicable to renal nutrition.

Outstanding Service Award
• Each year RPG selects one member with the OSA for recognition of his or her contributions to the field of renal nutrition.

Looking for a resource? Suggestions or ideas?

We want to hear from you!
Melissa Prest, MS, RD, CSR, LDN
RPG Electronic Media Manager
mediamgr@renalnutrition.org
Eating Well

Eating well means enjoying food and living healthier, and is simple as 1, 2, 3!

1. start fresh
Choose fresh foods to boost fiber and flavor.

2. shop savvy
Read the labels to know what's in your food.

3. cook from scratch
Make the most of your time and money.

Find the “PHOS”
Look for “phos” in the list of ingredients, and avoid added phosphates by comparing similar products.
1. **Start Fresh:**
   - Choose fresh fruits and vegetables to get the most fiber.
   - Select fresh meats, fish, and poultry without added breading or seasonings.
   - Go for plain whole grains, beans, rice, and pasta. Avoid added salt.
   - If fresh foods are not available, choose freshly frozen options.

2. **Shop Savvy:**
   - Opt for whole grains and 100% whole-grain products when available.
   - Avoid added salt and choose foods low in sodium.
   - Buy local in-season produce for a better value.
   - Check local ads and coupons.
   - Choose all-natural meats when possible.
   - Avoid foods with added phosphates.

3. **Cook from Scratch:**
   - Try simple recipes that appeal to you!
   - For big flavors, try salt-free herbs and spices, garlic, onions, peppers, vinegar, or lemon and lime.
   - Limit fat by grilling, baking, boiling, steaming, or broiling.
   - Cook from scratch even if you only have a hot-plate or microwave.
   - Make enough for leftovers, and freeze them in single-serving, easy to reheat containers.
   - Ask your dietitian for more tips on how to cook affordably and enjoyably.
   - Cooking with family or friends is quality time, and it’s a great motivator for cooking from scratch.

**Start fresh, shop savvy, and cook from scratch:** three things that will help you save money and create more healthy meals.

One change I would like to make is ______________________________.
INDICATION
Velphoro® (sucroferric oxyhydroxide) is a phosphate binder indicated for the control of serum phosphorus levels in patients with chronic kidney disease on dialysis.

IMPORTANT SAFETY INFORMATION
• Velphoro must be administered with meals. Velphoro tablets must be chewed and not swallowed whole. To aid with chewing and swallowing, the tablets may be crushed.
• Patients with peritonitis during peritoneal dialysis, significant gastric or hepatic disorders, following major gastrointestinal (GI) surgery, or with a history of hemochromatosis or other diseases with iron accumulation have not been included in clinical studies with Velphoro. Monitor effect and iron homeostasis in such patients.
• In a parallel design, fixed-dose study of 6 weeks duration, the most common adverse drug reactions to Velphoro chewable tablets in hemodialysis patients included discolored feces (12%) and diarrhea (6%).
• Velphoro can be administered concomitantly with oral calcitriol, ciprofloxacin, digoxin, enalapril, furosemide, HMG CoA reductase inhibitors, hydrochlorothiazide, losartan, metoprolol, nifedipine, omeprazole, quinidine and warfarin. Take doxycycline at least 1 hour before Velphoro. Velphoro should not be prescribed with oral levothyroxine.

Please see Brief Summary on adjacent page or visit www.Velphoro.com for full Prescribing Information.

† A 52-week, open-label, active-controlled, phase 3 study evaluated the safety and efficacy of Velphoro in lowering serum phosphorus levels in patients (N=1,054) with chronic kidney disease on hemodialysis or peritoneal dialysis. 1


Velphoro is a registered trademark of Vifor Fresenius Medical Care Renal Pharma Ltd.
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INDICATIONS AND USAGE
Velphoro (sucroferric oxyhydroxide) is a phosphate binder indicated for the control of serum phosphorus levels in patients with chronic kidney disease on dialysis.

DOSAGE AND ADMINISTRATION
Velphoro tablets must be chewed and not swallowed whole. To aid with chewing and swallowing, tablets may be crushed.

The recommended starting dose of Velphoro is 3 tablets (1,500 mg) per day, administered as 1 tablet (500 mg) 3 times daily with meals. Adjust by 1 tablet per day as needed until an acceptable serum phosphorus level is reached, with regular monitoring afterwards. Titrate as often as weekly.

DOSAGE FORMS AND STRENGTHS
Velphoro (sucroferric oxyhydroxide) chewable tablet 500 mg.

CONTRAINDICATIONS
None.

WARNINGS AND PRECAUTIONS
Patients with peritonitis during peritoneal dialysis, significant gastric or hepatic disorders, following major gastrointestinal surgery, or with a history of hemochromatosis or other diseases with iron accumulation have not been included in clinical studies with Velphoro. Monitor effect and iron homeostasis in such patients.

ADVERSE REACTIONS
In a parallel design, fixed-dose study of 6 weeks duration, the most common adverse drug reactions to Velphoro chewable tablets in hemodialysis patients included discolored feces (12%) and diarrhea (6%).

To report SUSPECTED ADVERSE REACTIONS, contact Fresenius Medical Care North America at 1-800-323-5188 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

DRUG INTERACTIONS
Velphoro can be administered concomitantly with oral calcitriol, ciprofloxacin, digoxin, enalapril, furosemide, HMG-CoA reductase inhibitors, hydrochlorothiazide, losartan, metoprolol, nifedipine, omeprazole, quinidine and warfarin.

Take doxycycline at least 1 hour before Velphoro.

Velphoro should not be prescribed with oral levothryoxine.

USE IN SPECIFIC POPULATIONS
Pregnancy
Pregnancy Category B: Reproduction studies have been performed in rats and rabbits at doses up to 16 and 4 times, respectively, the human maximum recommended clinical dose on a body weight basis, and have not revealed evidence of impaired fertility or harm to the fetus due to Velphoro. However, Velphoro at a dose up to 16 times the maximum clinical dose was associated with an increase in post-implantation loss in pregnant rats. Animal reproduction studies are not always predictive of human response. There are no adequate and well-controlled studies in pregnant women.

Labor and Delivery
No Velphoro treatment-related effects on labor and delivery were seen in animal studies with doses up to 16 times the maximum recommended clinical dose on a body weight basis. The effects of Velphoro on labor and delivery in humans are not known.

Nursing Mothers
Since the absorption of iron from Velphoro is minimal, excretion of Velphoro in breast milk is unlikely.

Pediatric Use
The safety and efficacy of Velphoro have not been established in pediatric patients.

Geriatric Use
Of the total number of subjects in two active-controlled clinical studies of Velphoro (N=835), 29.7% (n=248) were 65 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects.

OVERDOSAGE
There are no reports of overdosage with Velphoro in patients. Since the absorption of iron from Velphoro is low, the risk of systemic iron toxicity is low. Hypophosphatemia should be treated by standard clinical practice.

Velphoro has been studied in doses up to 3,000 mg per day.

HOW SUPPLIED/STORAGE AND HANDLING
Velphoro are chewable tablets supplied as brown, circular, bi-planar tablets, embossed with “PA 500” on 1 side. Each tablet of Velphoro contains 500 mg iron as sucroferric oxyhydroxide. Velphoro tablets are packaged as follows:

NDC 49230-645-51 Bottle of 90 chewable tablets

Storage
Store in the original package and keep the bottle tightly closed in order to protect from moisture.

Store at 25°C (77°F) with excursions permitted to 15 to 30°C (59 to 86°F).

PATIENT COUNSELING INFORMATION
Inform patients that Velphoro tablets must be chewed and not swallowed whole. To aid with chewing and swallowing, the tablets may be crushed [see Dosage and Administration].

Velphoro should be taken with meals.

Instruct patients on concomitant medications that should be dosed apart from Velphoro [see Drug Interactions].

Inform patients that Velphoro can cause discolored (black) stool.

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920 Winter Street
Waltham, MA 02451

US Patent Nos. 6174442 and pending, comparable and/or related patents.

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### October 2017

**FNCE® 2017 Food & Nutrition Conference & Expo**  
October 21-24, 2017  
McCormick Place West  
Chicago, IL  

**ASN Kidney Week**  
October 31-November 5, 2017  
Morial Convention Center  
New Orleans, LA  
https://www.asn-online.org/education/kidneyweek/archives/future.aspx

### November 2017

**Board Certification as Specialist in Renal Nutrition Examination**  
Exam Window: November 1-21, 2017  

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### August 2017

**International Congress on Nutrition and Metabolism in Renal Disease 2017**  
August 2-5, 2017  
Bangkok, Thailand  
http://www.ishd2017.org/index/ishd_Welcome.php

**NATCO 42nd Annual Meeting**  
August 2-5, 2017  
St. Louis Union Station Hotel  
St. Louis, MO  
http://www.natco1.org/Education/annual-meeting.asp

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Whether addressing nutrition questions from clients, consumers, students, or others, this is the ultimate resource for communicating science-based advice and answers on a myriad of topics. The latest edition has been completely updated to reflect the 2015-2020 Dietary Guidelines for Americans, current Academy positions, and the most up-to-date and authoritative public health guidelines.

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The Renal Dietitians Practice Group continues to explore the realm of social media and is now on Facebook and Twitter platforms. “Like” our Facebook page (Renal Dietitians-RPG, www.facebook.com/renaldietitians) and “follow” us on Twitter (Renal Dietitians-RPG, @RenalRDNs) for instant access to the most current information and resources in the field of renal nutrition including the following:

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- Kidney-Friendly Recipes and Demonstrations
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- Renal Nutrition Topics in Current News
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- …and much more!

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Renal Nutrition Forum Submission

Renal Nutrition Forum Submission

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We are always looking for articles about successful programs, research interventions, evaluations and treatment strategies and educational materials. Please forward information to: Managing Editor at rpgforumeditor@renalnutrition.org.

All submissions for publication should be submitted to the editor as an email attachment (MS Word file). Accepted articles from the Renal Nutrition Forum will be posted on the Members Only Section of the RPG website. Thus, please include a brief introduction or abstract plus 2-3 key words with article submissions.

Article Length:
Article length is determined by the Editor for each specific issue. The feature and advances in practice article (including abstract) is approximately 2500 words. Other supportive articles are 1000-1500 words; member highlights and reports are approximately 400-500 words.

Text Format:
Times New Roman font, 12 point, double space.

Tables/Illustrations:
Tables should be self-explanatory. All diagrams, charts and figures should be camera-ready. Each should be accompanied by a title and brief explanatory caption.

References:
References should be cited in the text in consecutive order parenthetically. At the end of the text, each reference should be listed in order of citation. The format should be the same as the Journal of the Academy of Nutrition and Dietetics.

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- Book:

- Chapter in a Book:

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  List author with first name, middle initial (if any), last name, professional suffix and affiliation below the title of the article. Also include the primary author’s complete contact information including affiliation, city, state and email address.
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